complications, necrotizing vasculitis and exacerbation of a pre-existing psoriasis, after use of G-CSF for treatment of a small cell lung cancer.

A 45-year-old woman was treated for a limited small cell lung cancer with a chemotherapy regimen consisting of cyclophosphamide (1 g m^{-2}), doxorubicin (50 mg m^{-2}), cisplatin (100 mg m^{-2}), and etoposide (200 mg m^{-2}). Because the patient had febrile neutropenia after the first course of chemotherapy, she was treated with 300 μg day^{-1} of G-CSF subcutaneously on days 3–10 following the end of the second course of chemotherapy. Four days later, she developed palpable purpuric lesions on the legs characteristic of a necrotizing vasculitis, and we observed exacerbation of psoriasis over thorax and legs. No biopsy was done as clinical aspects were rather characteristic.

There were no antibodies detected for nuclear cytoplasm antigens, hepatitis B and C viruses or human immunodefiency virus. Cryoglobulinemia and proteinuria were absent. The leucocyte count was $3{\cdot}5 \times 10^9\,l^{-1}$ with $2{\cdot}8 \times 10^9\,l^{-1}$ neutrophils; platelets were $211 \times 10^9\,l^{-1}$. Treatment with topical corticosteroid resolved psoriasis within 10 days, and purpuric lesions disappeared in about the same time. Considering the relative severity of the cutaneous lesions, the patient did not receive further G-CSF. Two additional courses with cisplatin and etoposide were administered simultaneously with thoracic radiotherapy, without recurrence of the cutaneous adverse effects.

Large reports of G-CSF treated patients with small cell lung cancer have been reported without cutaneous adverse effects (1,2). However, one patient with small cell lung cancer developed pyoderma gangraenosum during G-CSF therapy, and another patient showed necrotizing vasculitis with granulocyte-macrophage stimulating factor (3,4). Six other cases of cutaneous vasculitis have been reviewed in patients with various tumours, treated with leucocyte colony-stimulating factors. The eruptions are unrelated to the primary diseases and developed after 1 or 2 weeks of therapy. It is interesting that exacerbation of psoriasis has been noted in a patient with aplastic anemia subsequent to granulocyte macrophage-stimulating factor (5). To our knowledge, our case report is the first with both vasculitis and psoriasis. The mechanisms by which G-CSF exacerbate pre-existing psoriasis or induce vasculitis remain unknown. Various cytokines may be an indirect cause (tumour necrosis factor, interleukin-1) (6). We conclude that patients and physicians should be aware of the development of various inflammatory processes during G-CSF treatment. Patients with pre-existing psoriasis should be treated with caution, while receiving G-CSF.

L. J. Couderc*, B. Philippe*, N. Franck†,
E. Balloul-Delclaux* and
M. Lessana-Leibowitch†
**Service de Pneumologie*
Hopital Foch
Suresnes
†*Service de Dermatologie*
Hopital Tarnier
Paris, France.
14 September 1994

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Dear Editor

Gallbladder wall thickening as a sign of pulmonary embolism

Alvarez-Sala *et al.* described a patient with acute pulmonary embolism, in whom thickening of the gallbladder wall was shown by abdominal sonography (1). The authors suggest that this could be an initial sign of increased cystic and systemic venous hypertension, due to right cardiac failure, and hence to acute pulmonary embolism. The validity of the sign, however, is not established, since other cases have not been reported (2). We wish to report a case of acute pulmonary embolism we have observed, in which this sign was present.

A 92-year-old woman was admitted because of vomiting and tachycardia of several days duration. She denied dyspnoea and thoracic or abdominal pain. The remaining history was unremarkable. Blood pressure was 120/90 mmHg, heart rate was

84 beats min^{-1}, and respiratory rate was 20 breaths min^{-1}. The findings from cardiopulmonary and abdominal examination were normal. There were no signs of deep venous thrombosis in the lower extremities. The aspartate aminotransferase was 900 IU l^{-1}, the lactate dehydrogenase was 2524 IU l^{-1}, the γ-glutamyl-transpeptidase was 68 IU l^{-1}, the alkaline phospatase was 196 IU l^{-1}, and the total bilirubin was 1·03 mg dl^{-1}. A cholecystitis was suspected and abdominal sonography was performed, which demonstrated marked thickening of the gallbladder wall, with double appearance, and some hypoechoic lamellae within the wall. These signs are considered to be very suggestive of acute acalculous cholecystitis (3). Arterial gasometry revealed a PO_2 of 67·2 mmHg and a PCO_2 of 24·8 mmHg, with an alveoloarterial oxygen gradient of 48·8. The chest radiograph was normal, and an ECG showed sinus rhythm, right atrial enlargement, and an incomplete right bundle branch block. Since the patient did not show clinical signs of acute cholecystitis, we hypothesized a pulmonary embolism. There were biochemical signs of consumption of coagulation factors (thrombocytopenia, prolonged PT and PTT, reduced fibrinogen level, and elevated fibrin degradation products). Pulmonary perfusion scintigraphy showed some segmental defects. Treatment with heparin was started. Three weeks later, biochemical data and gas analysis were normal, and the abdominal sonogram indicated that the gallbladder wall was normal.

Thickening of the gallbladder wall is a sign suggestive of cholecystitis, but it has poor specificity, because there are many causes of gallbladder thickening. Oedema associated with congestive heart failure results in an absolutely identical appearance to that of inflammation (3). In cases of pulmonary embolism, the thickening of the gallbladder wall may be due to right cardiac failure and increased venous pressure. Our case confirms that reported by Alvarez-Sala *et al.* (1). Therefore, we think that the thickening of the gallbladder wall could be an initial sign of pulmonary embolism, although unusual. The presence of this sign in pulmonary embolism must be considered in order to avoid wrong diagnoses. However, its frequency needs further study.

V. Foresti
III Medical Department
Fatebenefratelli Hospital
Milan
Italy
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